The correspondence section is a public forum and, as such, is not peer-reviewed. EHP is not responsible for the accuracy, currency, or reliability of personal opinion expressed herein; it is the sole responsibility of the authors. EHP neither endorses nor disputes their published commentary.

ADHD, Lead, and PCBs: Appropriate **Comparison Studies**

doi:10.1289/ehp.1103513

In their article "Lead and PCBs as Risk Factors for Attention Deficit/Hyperactivity Disorder" (ADHD), Eubig et al. (2010) offered a large compilation of human and animal research supporting a relationship between these environmental contaminants and ADHD occurrence. Key to understanding such a relationship, however, is research quality, not quantity.

As Eubig et al. (2010) noted, ADHD is highly heritable, a history of ADHD in a parent or sibling being a strong predictor of ADHD occurrence in a child (Faraone and Doyle 2001). A sound study of the disorder and lead or polychlorinated biphenyls (PCBs) would therefore control for family history. The authors listed seven studies of lead exposure and ADHD in their Table 2, but five of the studies had no information on family history so they could not answer the question of a relationship. Another study suffered from likely underascertainment of parental history; even so, it remained significantly (p < 0.01) associated with ADHD in case children (Wang et al. 2008). The last study controlled for familial neuropsychiatric disease and reported no significant association of children's blood lead levels (BLLs) and ADHD, despite its ample cohort size of ≥ 1,700 (Ha et al. 2009).

In their Table 1, Eubig et al. (2010) listed 12 studies of human lead exposure and performance on test functions impaired in ADHD. Only 3 of the studies considered heritability as a possible confounder of this relationship, but none reported an association with performance (Chiodo et al. 2004, 2007; Stewart et al. 2006). This is surprising, given the marked heritability of ADHD, and raises the question of how well individual test functions may control for or serve as surrogates of ADHD diagnosis per se. Also, Stewart et al. (2006) found only a marginal (p < 0.047) association with medical record information on postnatal BLL in a potentially biased 60.9% of subjects, and no association (p < 0.641) with umbilical cord BLL in 88.6% of subjects.

According to National Health and Nutrition Examination Survey (NHANES) data, the proportion of elevated BLLs (≥ 10 μ g/dL) in U.S. children 1–5 years of age dropped from 77.8% in 1976-1980 to 0.9% in 2005-2008 (Centers for Disease Control and Prevention 2005; HealthyPeople.gov 2011). However, the occurrence of ADHD and its diagnostic predecessors has been rising

since the 1980s, if not before, offering no support for a positive association of BLL with ADHD (Pastor and Reuben 2008).

The PCB literature Eubig et al. (2010) presented in their Table 4 provided a picture little different from that of lead. PCB exposure is also apparently trending downward (Tee et al. 2003).

The dearth of well-controlled studies leaves open Eubig et al.'s question whether lead or PCBs exert an effect on ADHD occurrence beyond that exerted by heritability. This question cannot be answered satisfactorily until researchers consistently impose adequate control in their studies and funding agencies consistently require such control in the research they support.

The author has no actual or potential competing financial interests.

Jack Brondum

Epidemiology and Environmental Health Hennepin County Department of Human Services and Public Health Hopkins, Minnesota

E-mail: jack.brondum@co.hennepin.mn.us

REFERENCES

Centers for Disease Control and Prevention, 2005, Blood lead levels - United States, 1999-2002. MMWR Morbid Mortal Wkly Rep 54:513-516.

Chiodo LM, Covington C, Sokol RJ, Hannigan JH, Jannise J, Ager J, et al. 2007. Blood lead levels and specific attention effects in young children. Neurotoxicol Terat 29:538-546.

Chiodo LM, Jacobson SW, Jacobson JL. 2004. Neurodevelopmental effects of postnatal lead exposure at very low levels. Neurotoxicol Terat 26:359-371.

Eubig PA, Aguiar A, Schantz SL. 2010. Lead and PCBs as risk factors for attention deficit/hyperactivity disorder. Environ Health Perspect 118:1654-1666.

Faraone SV, Doyle AE. 2001. The nature and heritability of attention deficit/hyperactivity disorder. Child Adolesc Psychiatr Clin N Am 10:299-316.

Ha M, Kwon HJ, Lin MH, Jee YK, Hong YC, Leem JH, et al. 2009. Low blood levels of lead and mercury and symptom of attention deficit hyperacticvity in children: a report of the Children's Health and Environment Research (CHEER). NeuroToxicology 30:31-36.

HealthyPeople.gov. 2011. Healthy People 2020 Summary of Objectives. Available: http://www.healthypeople. gov/2020/topicsobjectives2020/pdfs/EnvironmentalHealth. pdf [accessed 28 January 2011].

Pastor PN, Reuben CA. 2008. Diagnosed Attention Deficit Hyperactivity Disorder and Learning Disability: United States, 2004-2006. Vital Health Stat Series 10 No. 237. Hvattsville, MD:National Center for Health Statistics. Available: http://www.cdc.gov/nchs/data/series/sr 10/ sr10_237.pdf [accessed 28 January 2011].

Stewart PW, Sergeant DM, Reihman J, Gump BB, Lonky E, Darvill T. 2006. Response inhibition during differential reinforcement of low rates (DRL) schedules may be sensitive to low-level polychlorinated biphenyl, methylmercury, and lead exposure in children. Environ Health Perspect 114:1923-1929.

Tee PG, Sweeney AM, Symanski E, Gardiner JC, Gasior DM, Schantz S. 2003. A longitudinal examination of factors related to changes in serum polychlorinated biphenyl levels. Environ Health Perspect 111:720-727.

Wang HL, Chen XT, Yang B, Ma FL, Wang S, Tang ML, et al.

2008. Case-control study of blood lead levels and attention deficit hyperactivity disorder in Chinese children. Environ Health Perspect 116:1401-1406.

ADHD, Lead, and PCBs: Eubig et al. Respond

doi:10.1289/ehp.1103513R

In response to Brondum's comments we would like to reiterate that the main purpose of our review (Eubig et al. 2010) was to examine the parallels between cognitive domains affected in children with attention deficit/hyperactivity disorder (ADHD) and domains shown to be affected in human and animal studies of developmental exposure to lead and polychlorinated biphenyls (PCBs), two environmental contaminants for which a relatively large body of literature exists. In doing so we hoped to explore the possible role of exposure to environmental contaminants in the variable phenotypic expression of ADHD, and to stimulate interest in further research in

In our review, we did not seek to identify individual behavioral tests or functional domains that could serve as surrogates for ADHD diagnosis. Nor did we make the case that developmental lead and PCB exposure are responsible for the rise in ADHD diagnoses in recent years. To the contrary, in our section on other environmental contaminants we specifically highlighted the fact that PCB and lead exposures are declining, whereas exposures to other chemicals—including brominated flame retardants, bisphenol A, phthalates, polyfluoroalkylated compounds and certain pesticides—are increasing. We note that studies of the potential role of these emerging contaminants in the etiology of ADHD are equally, if not more, important than further studies of lead and PCBs. In addition, there is a clear difference between exploring contaminants as potential contributors to ADHD risk as opposed to causing ADHD. Examining our Table 6 (Eubig et al. 2010), which showed a comparison of the strength of the evidence for cognitive domains affected in ADHD with domains affected in developmental lead and PCB exposure, should convince the reader that these three conditions are similar but not the same. Brondum seems to miss this point in implying that our review is without value because the studies that evaluated the association between lead and a diagnosis of ADHD, which comprise a relatively small part of our review, are flawed in his opinion.

No one is debating whether parental psychopathology should be considered as a possible confounding factor in studies that examine the association of contaminant exposure with specific neurobehavioral diagnoses, including ADHD. Braun et al. (2007) acquiesced to that point in a reply to the first of several letters to the editor by Brondum (2007) on the same topic. However, Braun et al. (2007) noted that including such information is not always possible when, for example, the use of National Health and Nutrition Examination Survey (NHANES) data or other constraints on study design do not allow it. Although we agree that failure to control for parental psychopathology is a weakness in many of the published studies reporting an association between childhood lead exposure and a diagnosis of ADHD, we believe that the consistency of the association across several published studies using different study designs adds to the weight of evidence that this is a real association and not a spurious effect due to uncontrolled confounding.

We hope that this reply clarifies the goals of our review for those faced with the challenge of assessing the neurobehavioral effects of emerging contaminants and their possible contribution to the phenotypic expression of ADHD or other neurodevelopmental disorders.

The authors declare they have no actual or potential competing financial interests.

Paul A. Eubig Andréa Aguiar Susan L. Schantz

Department of Comparative Biosciences College of Veterinary Medicine University of Illinois at Urbana-Champaign Urbana, Illinois E-mail: eubig@illinois.edu

REFERENCES

Braun JM, Lanphear BP, Kahn RS, Froehlich T, Auinger P. 2007. ADHD: Braun et al. Respond [Letter]. Environ Health Perspect 115:A399. .

Brondum J. 2007. Environmental Exposures and ADHD [Letter]. Environ Health Perspect 115:A398.

Eubig PA, Aguiar A, Schantz SL. 2010. Lead and PCBs as risk factors for attention deficit/hyperactivity disorder. Environ Health Perspect 118:1654–1666.

Home Energy-Efficiency Retrofits

doi:10.1289/ehp.10733

In the February 2011 issue of *EHP*, Manuel (2011) took an important look at some potential adverse health implications of home energy retrofits. Here, we further discuss the complexity of possible indoor environmental concerns and encourage incorporation of comprehensive homeowner education campaigns in weatherization programs.

The reduction of air infiltration by air sealing is a common energy retrofit measure (McCold et al. 2008). Several field studies of weatherized homes have reported average reductions in air leakage of 13–40% (Berry 1997; Judkoff et al. 1988), although the impact of weatherization on actual air

exchange rates and indoor pollutant concentrations is poorly understood. Moreover, studies have seldom evaluated the effects of weatherization on low-income groups or vulnerable populations (e.g., asthmatic or elderly), although occupants in low-income residences are at higher risk for many indoor environmental hazards (Evans and Kantrowitz 2002), and some population subgroups may also be disproportionately affected by indoor air pollution (Hun et al. 2009).

Although some research exists on the impact of weatherization on indoor concentrations of combustion products, radon, and moisture, other indoor pollutants deserve attention. For example, Logue et al. (2011) identified nine priority indoor air pollutant hazards in U.S. residences, which, among others, have been associated with a wide range of both chronic and acute health effects (e.g., benzene, 1,4-dichlorobenzene, formaldehyde, naphthalene, particulate matter < 2.5 μm in aerodynamic diameter). Moreover, reducing air exchange rates in residences will likely increase indoor concentrations of reactive pollutants and the probability of chemical reactions occurring between them indoors (Weschler and Shields 2000), generating by-products associated with respiratory symptoms and asthma, such as low-molecularweight aldehydes, dicarbonyls, and secondary organic aerosols (Jarvis et al. 2005). On the other hand, reductions in air infiltration should decrease penetration of outdoor pollutants, which is of particular importance in traditionally leakier low-income households (Chan et al. 2005) in neighborhoods with high outdoor air pollution. Thus, we urge the environmental health community to investigate the net effects of weatherization on a wide variety of indoor and outdoor pollutants and health outcomes.

Implementation of home energy retrofits also creates an opportunity to incorporate innovative, engaging homeowner education strategies to reduce both energy consumption and indoor environmental risks. Occupant behavior has a major influence on both energy consumption (Allcott and Mullainathan 2010) and indoor exposures to pollutants (Meng et al. 2005). Furthermore, many indoor air quality risks can be mitigated with relatively simple home behavior practices, such as using exhaust fans, avoiding toxic cleaning chemicals, and using appropriate air cleaners (Brugge et al. 2003). However, we have learned from research on household energy consumption that educational materials alone usually fail to alter behaviors (Charles 2009). Greater energy savings from home retrofits could be achieved by complementing homeowner education campaigns with regular feedback on energy use and economically motivational programs (Peschiera et al. 2010). Additionally, home walkthroughs with trained building specialists can identify energy-inefficient behaviors and appliances in conjunction with potential indoor environmental hazards. These and other behavior-change strategies to promote green and healthy housing should be made available to weatherization programs across the country, and their effectiveness should be assessed. Because home weatherization is currently a priority of the federal government, this is a crucial time to address these fundamental research questions and implement the findings nationwide.

All authors are current or former trainees or affiliates of a National Science Foundation IGERT program in Indoor Environmental Science and Engineering at The University of Texas at Austin (award DGE 0549428).

M.C.J. is employed by Lennox International, Inc., which produces HVAC systems and products. The other authors declare they have no actual or potential competing financial interests.

Brent Stephens Ellison M. Carter Elliott T. Gall C. Matt Earnest Elizabeth A. Walsh

National Science Foundation Integrative Graduate Education and Research Traineeship (IGERT) Program in Indoor Environmental Science and Engineering The University of Texas at Austin Austin, Texas

E-mail: stephens.brent@mail.utexas.edu

Diana E. Hun

Oak Ridge National Laboratory Oak Ridge, Tennessee

> Mark C. Jackson Lennox International Inc. Carollton, Texas

REFERENCES

Allcott H, Mullainathan S. 2010. Behavior and energy policy. Science 327(5970):1204–1205.

Berry LG, Brown MA. 1994. Patterns of Impact in the Weatherization Assistance Program: A Closer Look. ORNL/ CON-331. Oak Ridge, TN:Oak Ridge National Laboratory. Available: http://weatherization.ornl.gov/pdfs/ORNL_CON-331.odf [accessed 8 June 2011].

Brugge D, Vallarino J, Ascolillo L, Osgood ND, Steinbach S, Spengler J. 2003. Comparison of multiple environmental factors for asthmatic children in public housing. Indoor Air 13(1):18–27.

Chan W, Nazaroff W, Price P, Sohn M, Gadgil A. 2005. Analyzing a database of residential air leakage in the United States. Atmos Environ 39(19):3445–3455.

Charles D. 2009. Leaping the efficiency gap. Science 325(5942):804-811.

Evans GW, Kantrowitz E. 2002. Socioeconomic status and health: the potential role of environmental risk exposure. Annu Rev Public Health 23(1):303–331.

Hun DE, Siegel JA, Morandi MT, Stock TH, Corsi RL. 2009. Cancer risk disparities between Hispanic and non-Hispanic white populations: the role of exposure to indoor air pollution. Environ Health Perspect 117:1925–1931.

Jarvis J, Seed M, Elton R, Sawyer L, Agius R. 2005. Relationship between chemical structure and the occupational asthma